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NIH Fiche to Paper Journal

TITLE:	RADIOLOGIC CLINICS OF NORTH AMERICA
PUBLISHER/PLACE:	Saunders. Philadelphia, London,
VOLUME/ISSUE/PAGES:	1993 Jan;31(1):121-34 121-34
DATE:	1993
AUTHOR OF ARTICLE:	Mohammadi-Araghi H; Haery C
TITLE OF ARTICLE:	Fibro-osseous lesions of craniofacial bones. The r
ISSN:	0033-8389
OTHER NOS/LETTERS:	Library reports holding volume or year
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FIBRO-OSSEOUS LESIONS OF CRANIOFACIAL BONES

The Role of Imaging

Hossein Mohammadi-Araghi, DDS, MS, and Cameron Haery

Fibro-osseous lesions of the craniofacial bones are a challenging group of pathologic conditions that are difficult to classify and treat.[36] Reports in literature made over the last four decades indicate several semantic difficulties with these lesions.[29] Unfortunately, there are no universally accepted clinical, radiographic, or histopathologic criteria that categorically distinguish these lesions from one another.[29] A common denominator to all is the replacement of the bone with a benign fibrous tissue containing various amounts of mineralized (calcified) structures.[36] It is often quite a challenge to establish a reasonable biologic behavior for these disorders in terms of whether the lesions in question are truly neoplastic, developmental, or reactive processes. Fibro-osseous lesions of the jaw may be divided into two categories: fibrous dysplasia, a developmental lesion due to idiopathic arrest in the normal maturation of bone at the woven bone stage, and those that have been postulated to originate from the periodontal ligament (the portion of periodontium which attaches the root of a tooth to the alveolus).[43, 44] Lesions in the latter group include ossifying fibroma, cementifying fibroma, cemento-ossifying fibroma, and periapical cemental dysplasia. The criteria for classification of these lesions are not well defined and are variable among institutions and authorities. Margo et al[29] have offered the following classifications and guidelines for a better understanding of the various fibro-osseous lesions:

Fibrous dysplasia. Fibrous dysplasia is a lesion composed of curvilinear woven bone trabeculae that appear unconnected in a single section and emerge from a whorled spindle-cell background. Osteoblasts are typically absent except near the area of transition to normal bone.

Ossifying fibroma. An ossifying fibroma is a predominantly fibrous lesion with variable amounts of woven and lamellar bone trabeculae that appear interconnected in a single section. Plump osteoblasts may rim trabeculae.

Psammomatoid (juvenile) ossifying fibroma (active juvenile ossifying fibroma). This lesion is composed of numerous small rounded mineralized collagenous foci (psammomatoid ossicles) embedded within a relatively cellular stroma consisting of small uniform stellate and spindle-shaped cells without notable mitotic activity.

Cementifying fibroma. Numerous round "cementicles" embedded within a benign spindle cell stroma is called a cementifying fibroma. Although its histologic pattern is similar to pasammomatoid ossifying fibroma, the term should be restricted to those lesions arising in relation to tooth structures.

Cemento-ossifying fibroma. This is a jaw lesion in which clear-cut fibrous dysplasia or ossifying fibroma coexists with the small round cementicle pattern of cementifying fibroma.

Osteoma. An osteoma is a well-differentiated benign tumor consisting predominantly of thick

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lamellar bone; it typically is seen in frontal and ethmoid sinuses. Microscopically, osteomas can be divided into three general groups that may overlap: ivory, mature, and fibrous.[39] The ivory osteoma is composed mostly of irregular bony trabeculae and a minimal amount of fibrous connective tissue. The mature osteoma has thinner trabeculae and more intertrabecular fibrous tissue. The fibrous (spongiose) osteoma has fibrous tissue and osteoblastic activity. The fibrous osteoma is similar histopathologically to the ossifying fibroma, but it does not exhibit the locally aggressive behavior characteristic of the ossifying fibroma.[39]

Osteoblastoma. Osteoblastoma is a neoplasm consisting of plentiful amounts of osteoid produced by overabundant osteoblasts, which, although benign, vary considerably in size, shape, and staining qualities. Osteoclasts are numerous and the background is highly vascular.[29]

FIBROUS DYSPLASIA

Fibrous or fibro-osseous dysplasia is somewhat more common than other fibro-osseous lesions discussed herein. It is a developmental disorder of a growing bone for which the etiology is unknown. Fibrous dysplasia is considered by some authors to be a hamartomatous malformation that presumably results from an idiopathic arrest in the maturation of bone at the woven bone stage.[39] In this condition, the modular portion of a normal bone is gradually being replaced by an abnormal fibroconnective tissue proliferation containing various amounts of osteoid and osseous structures, presumably of metaplastic nature. The bone trabeculae remain, for the most part, at the woven stage (an early stage of bone formation while collagen fibers are arranged haphazardly) and usually do not mature to lamellar (collagen fibers are paralleled) state of development. Seventy-four percent of the benign fibro-osseous lesions seen in the gnathic bones among Africans are reported to be fibrous dysplasia.[1, 2, 13, 34] The lesion may involve one skeletal bone (monostotic) or several bones concomitantly (polyostotic). Most often, this lesion is monostotic fibrous dysplasia (70%).[21, 22, 32, 35] The polyostotic type account for 30% of cases of fibrous dysplasia.[4, 8, 9, 16, 18, 28, 37] A monostotic fibrous dysplasia does not progress to the polyostotic form of the disease. The process may involve the facial bones, skull bones, or both.[26] Polyostotic fibrous dysplasia may be associated with endocrinopathies, which account for 3% to 5% of all cases. This rare form of the disease, accompanied by skin pigmentations (café au lait spots) ipsilateral to osseous involvement and precocious puberty in girls, is referred to as Albright's syndrome. Many other endocrine disorders, however, also have been identified, including hyperparathyroidism, Cushing's syndrome, hyperthyroidism, and acromegaly as well as adenomas of various endocrine glands.[3, 5, 7, 23, 25] Monostotic fibrous dysplasia commonly occurs in the rib (24%), femur (17%), tibia (13%), mandible (12%), and maxilla (12%). In the skull, fibrous dysplasia commonly involves the ethmoid, sphenoid, frontal, and temporal bones in decreasing order, respectively.

Some authorities postulate that skeletal lesions encountered in fibrous dysplasia are nonneoplastic and hamartomatous growth.[39] Grossly fibrous dysplasia is a firm, gritty, noncapsulated lesion.[39] Histologically, fibrous dysplasia is characterized by a moderately cellular fibrous stroma with uniform, benign-appearing spindle cells. Integrated throughout the fibrous stroma are the foci of irregularly shaped trabeculae of immature woven bone. The collagen fibers may have a haphazard arrangement or may be arranged in a whorled pattern. The fibroblasts are well-differentiated and mature; there is no mitotic activity.[6] Ossification is irregular, and most of the trabeculae do not have osteoblasts around them. The absence of osteoblasts helps to distinguish fibrous dysplasia from ossifying fibroma.[39] The trabeculae appear similar to Chinese script and have been referred to as a "Chinese character trabeculae."[36]

Clinical Features

Clinical presentation of fibrous dysplasia varies with the primary bone involved and the extent of the disease. For example, pain and tenderness are related to a pathologic fracture of weight-bearing bones, visual complications occur with sphenoethmoidal complex involvement, hearing disturbances are present when the temporal bone is affected, and displacement of the teeth is caused by jaw bone involvement. The most common clinical presentations of the disease, however, are facial asymmetry and an asymptomatic swelling. Fibrous dysplasia usually has its clinical onset during early life, usually late childhood or early

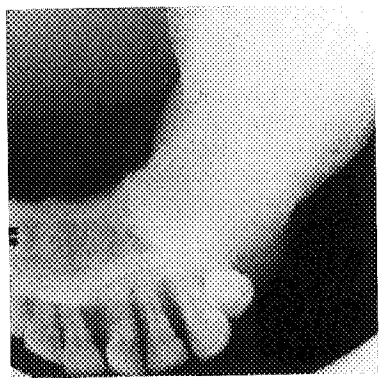

Figure 1. Occlusal view of left mandible of a 30-year-old woman with fibrous dysplasia. Note radiodense buccal and lingual mandibular expansile lesion with ground glass appearance.

adolescence. Patients with the polyostotic form of the disease are considerably younger. There is an equal sex distribution in monostotic fibrous dysplasia. The polyostotic type of the disease, with endocrine disorder, has a clear female sex predilection. Approximately one third of the patients who have fibrous dysplasia show an elevated serum alkaline phosphatase level.[24] Elevation of serum phosphatase is unrelated to the extent of the lesion or associated fracture.

Radiographic Features

Radiographic features of fibrous dysplasia vary depending on the stage of development and amount of bony matrix within the lesion. In general, changes in the involved bones range from lucent zones to large diffuse areas of sclerosis. In its early stage, the lesion appears radidolucent and may be relatively well defined. The lesion is often unilocular. As the process matures and metaplastic trabecular bone predominates, the radiolucent appearance of the early lesion changes into a smudged, mottled radio-opacity. Further increases in bone formation within the lesion create a radiographic appearance that is referred to as ground glass, frosted glass, or orange peel[13] (Fig. 1). Lack of a sharp marginal definition is a significant radiographic feature of fibrous dysplasia, especially during its late stages. The process appears to blend into the surrounding bone—to leave a lesion without readily discernible borders (Figs. 2*A* and 3*B*). The lamina dura of the teeth in the affected area may be completely effaced.[23] This type of radiographic feature is quite characteristic of maxillary fibrous dysplasia and strongly indicates the disease.[23] In general, radiographic features of the craniofacial fibrous dysplasia have been subclassified into three different patterns: the pagetoid (56%) (Fig. 2*A*), the radiolucent (cystic) type (21%) (Fig. 3*A*), and the sclerotic (23%) (Fig. 3*A*). Facial bones and the base of the skull are preferentially involved by the sclerotic form. The lytic form is often seen in calvarial bones. Maxillary and mandibular involvement may cause teeth impaction and dental malocclusion (see Fig. 1). Root resorption and loosening of the teeth, however, are most unusual. Fibrous dysplasia may displace the mandibular canal superiorly (Fig. 3*A*), which is a significant finding in differentiating this lesion from other fibro-osseous lesions in the mandible, as almost all other lesions in this group develop above the canal and close to dental structures (being dental-related lesions) (see Fig. 9*B*).[20] A wide variety of lesions should be considered in the differential diagnosis of fibrous dysplasia. A solitary lesion could mimic a tumor or tumorlike condition. Included in this group are nonossifying fibroma, ossifying fibroma, simple bone cyst, focal eosinophilic granuloma (Langerhans histiocytosis), aneurysmal bone cyst, giant cell reparative granuloma, giant cell tumor, osteoblastoma, enchondroma, and intraosseous he-

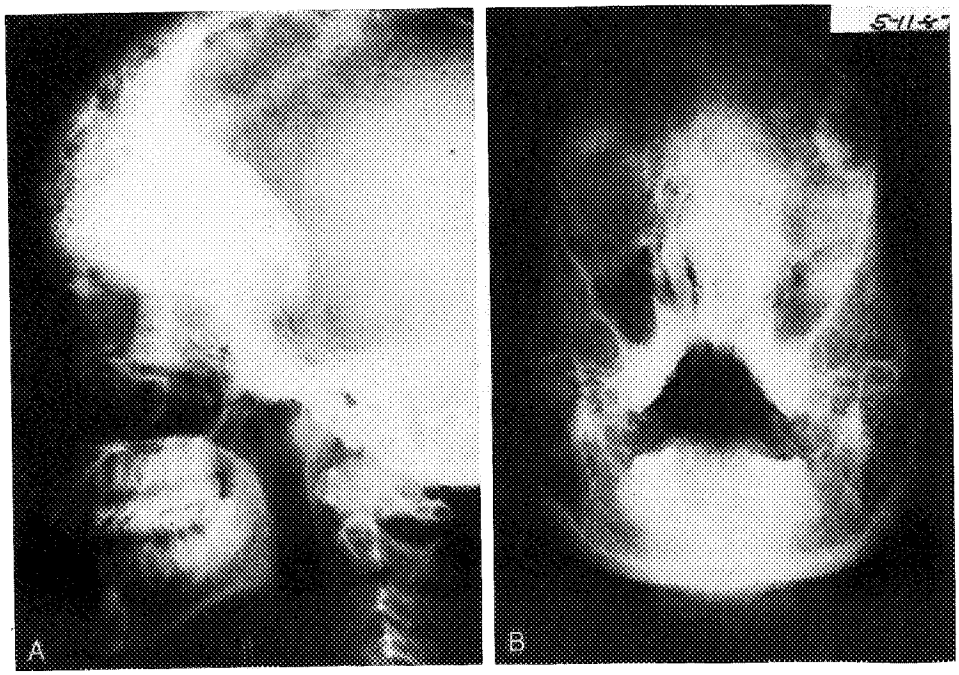

Figure 2. Craniofacial fibrous dysplasia (pagetoid type) in a 35-year-old man. *A*, Lateral skull view reveals expansion and sclerosis of the horizontal and vertical plates of frontal bone with areas of irregular as well as uniform bone formation. Note involvement of anterior clinoids. *B*, Waters' view showing complete obliteration of nasoethmoidal region caused by increased bone formation.

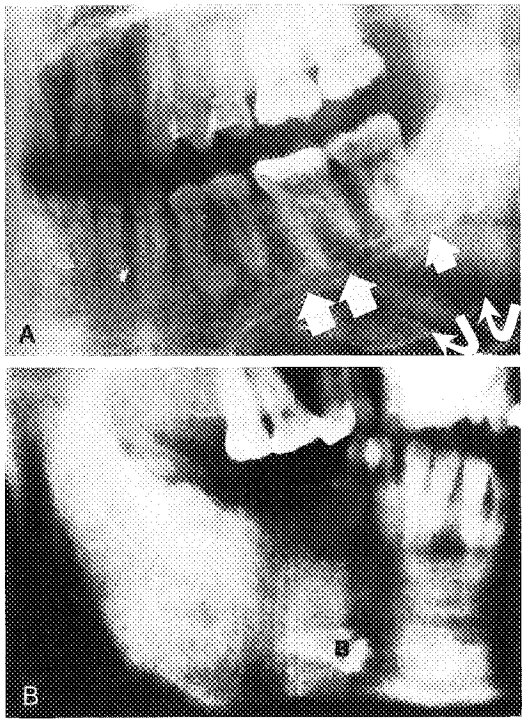

Figure 3. *A,* Mandibular fibrous dysplasia (cystic type). Note radiolucent expansile lesion of mandible *(curved arrows)* with superior displacement of the inferior alveolar canal *(straight arrows). B,* Late stage of a fibrous dysplasia with an impacted mandibular bicuspid *(B).* Note marked sclerosis and expansion of the body and ramus of the mandible. There is poor delineation between normal and abnormal bone.

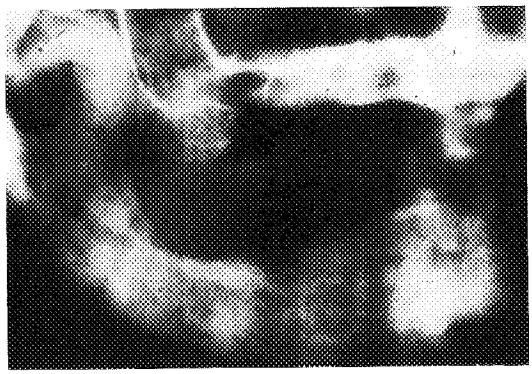

Figure 4. Chronic sclerosing osteitis. Panoramic mandibular view of a 57-year-old woman with a history of excruciating pain over the right mandible. Note, diffuse irregular patchy radiodensity of the right mandible. The medullary portion of the bone is partially obliterated.

mangioma. Among inflammatory conditions, chronic sclerosing osteitis (Fig. 4) and chronic granulomatous inflammatory reaction should also be included in the differential diagnosis. In polyostotic form, the differential diagnosis should include hyperparathyroidism, neurofibromatosis, multifocal eosinophilic granuloma, osteogenesis imperfecta, Paget's disease, and metastatic lesions. Fibrous dysplasia may be complicated by an aneurysmal bone cyst developing within the lesion and by malignant transformation, such as osteosarcoma and fibrosarcoma, especially of the craniofacial region.[14, 41]

Computed tomographic (CT) scanning has been useful in establishing the diagnosis of fibrous dysplasia and defining the extent of the lesion (Fig. 5).[10, 31] Involvement of the optic canals (Fig. 6), superior and inferior orbital fissures (Fig. 6*B*), frontonasal duct, natural openings of sphenoid sinuses, and ostiomeatal complex of the nasoethmoid region can best be evaluated by CT scanning (Figs. 5 and 6). CT characteristics of fibrous dysplasia include expansion of the involved bone with heterogeneous pattern of CT densities associated with scattered or confluent islands of bone formation (Figs. 5 and 6). The CT attenuation values have been reported to range from 34 to 513 Hounsfield units (HU) depending on the fibrous tissue and bone rate[45]; however, in some cases, it may exceed far more than 513 HU.[31]

On magnetic resonance (MR) imaging fibrous dysplasia exhibits an intermediate signal on T1-weighted and proton-weighted images and heterogeneous hypointense signal on T2-weighted scans (Fig. 7). After the intravenous infusion of paramagnetic gadolinium-diethylenetriamine pentaacetic acid (Gd-DTPA) contrast material, lesions of fibrous dysplasia will show a moderate to significant contrast enhancement on MR scans (Fig. 7*D*). It is exceedingly important to be familiar with the MR appearance of fibrous dysplasia, because its MR characteristics may be confused with many other benign and malignant lesions of the craniofacial structures.

FIBRO-OSSEOUS LESIONS OF PERIODONTAL LIGAMENT ORIGIN

Fibro-osseous lesions that arise from dental structures encompass a large variety of entities, varying from small, asymptomatic lesions to large, aggressive expansile lesions.[36] Oral pathologists have wrestled, as have head and neck pathologists, with a similar problem in trying to distinguish between cementifying fibroma and ossifying fibroma of the jaws.[15, 44] It has been proposed that the mesenchymal cells of the periodontal membrane (connective tissue that anchors the tooth to the alveolar ridge) are multipotential cells capable of differentiation into cementum, osteoid, or fibrous tissue.[29] This group of lesions includes ossifying fibroma, cementoma, cementifying fibroma, cemento-ossifying fibroma, and periapical cemental dysplasia.

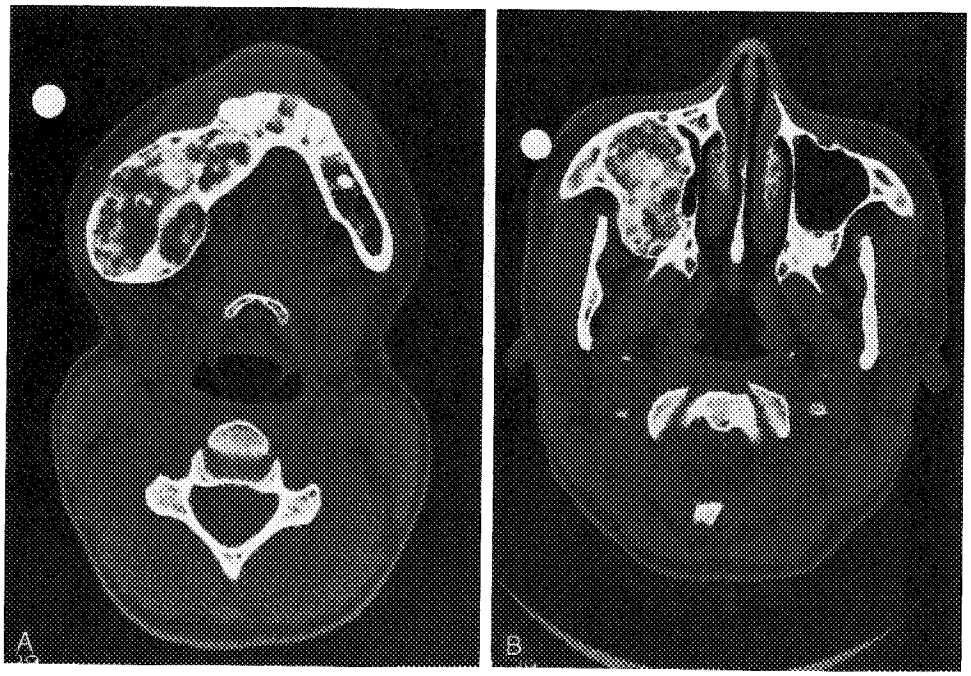

Figure 5. Fibrous dysplasia. *A*, CT scan shows marked expansion of right side of mandible with irregular islands of bone formation. *B*, CT scan shows obliteration of right maxillary antrum with areas of irregular bone formation.

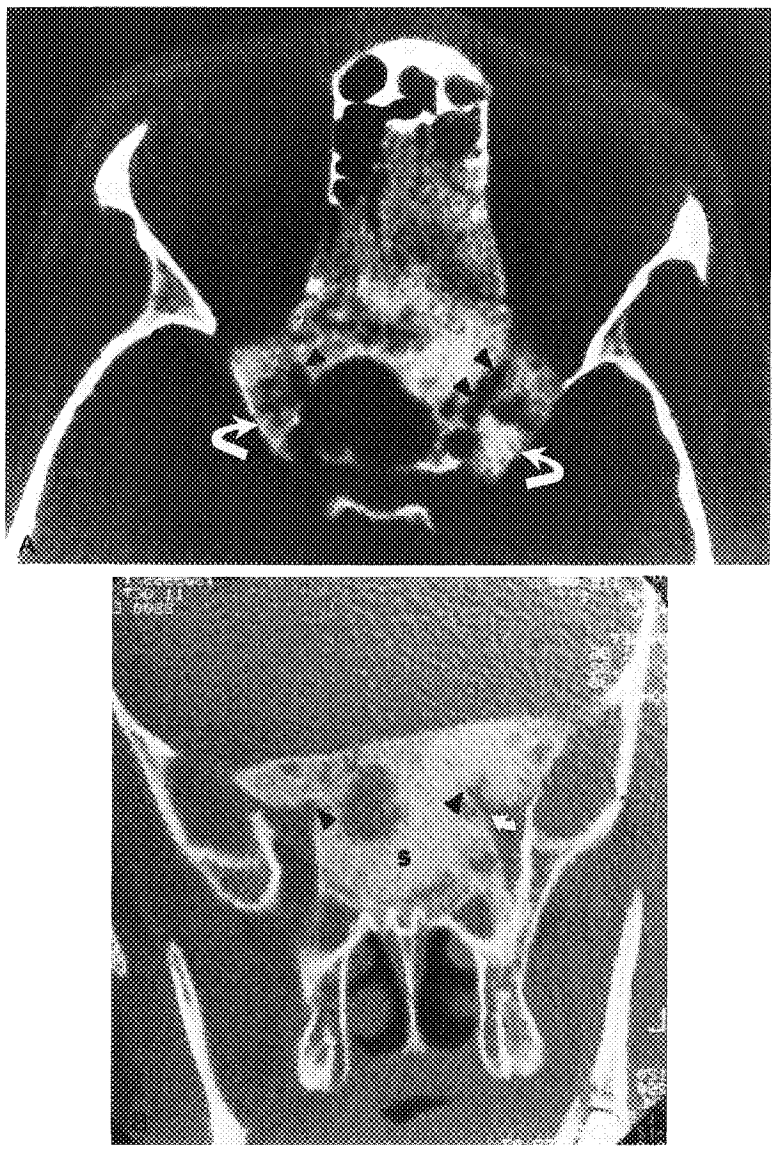

Figure 6. Fibrous dysplasia. *A,* Axial CT scan shows involvement of ethmoid air cells and lesser wings of sphenoid bone *(curved arrows).* Notice marked constriction of optic canals *(arrowheads). B,* Coronal CT scan shows involvement of sphenoid bone *(S),* lesser wings, constriction of optic canals *(arrowheads),* and superior orbital fissures *(curved arrow).* (Courtesy of M. Mafee, MD.)

Ossifying Fibroma

Ossifying fibroma is a benign, gradually expansile, and fairly encapsulated tumor predominantly of the jawbones. This tumor was reported as early as 1865 in the British literature. The term *ossifying fibroma* was coined by Montgomery.[33] The lesion has also been designated *fibrous osteoma* and *fibro-osteoma*.[15, 17] Except for the juvenile variety, ossifying fibromas seem to occur mostly in the third and fourth decade of life, with women more often than men affected.[44] The lesions usually arise in close proximity to the roots of the teeth. The most commonly involved bone appears to be the mandible, with a high affinity to the molar area. Cases of ossifying fibromas of the other craniofacial bones have been reported.[12, 27]

Histologically, ossifying fibroma consists of moderately cellular, delicately interlacing collagen fibers and usually well-vascularized stroma containing various amounts of calcified materials. Calcification may appear as irregular bony structures (woven or lamellar) and spicules. These spicules resemble psammoma bodies, an observation that has prompted some authorities to use the term *psammomatoid ossifying fibroma*.[29] Lamellar bone formation and osteoblastic rimming found in ossifying fibroma are believed by some pathologists to be differentiating features of fibrous dysplasia.[38]

A variant of ossifying fibroma, active juvenile ossifying fibroma, has been described in children. This tumor is a rapidly enlarging as well as destructive process, occurring predominantly in the maxilla of children and adolescents usually younger than the age of 15 years.[11, 19, 29, 40, 42] The relationship between active juvenile ossifying fibroma and ossifying fibroma, however, needs elucidation.

Radiographic Features. Ossifying fibroma, in its early stage, appears to be solitary, cystlike, and osteolytic, without a prominent periosteal reaction (Fig. 8*A*). At a later stage of maturation, lesions are radiopaque and surrounded by a uniform radiolucent rimming (Fig. 8*B*). Growth tends to be concentric within the medullary part of the bone with outward expansion approximately equal in all directions. Occasionally, a sclerotic border may separate the lesion from the adjacent normal bone. A thin shell of bone is usually found along the border of the tumor and extraosseous expansion is unusual. Displacement and destruction of the adjacent structures may occur as the tumor grows (Fig. 9). Teeth are displaced away from the lesion; root resorption may range from minimal to severe.

Differential Diagnosis. The differential diagnosis of ossifying fibroma should include any jawbone lesions whether radiolucent, radiopaque, or mixed type in radiodensities.

At the early stage of development, the lesion may appear as unilocular or multilocular radiolucencies, which may cause confusion with odontogenic tumors such as ameloblastomas, or odontogenic cysts such as keratocysts, or a simple bone cyst. When ossifying fibroma presents as a lesion with predominant radiopacity, other lesions, including sclerosing osteitis, osteoma, and osteoblastoma should be considered in the differential diagnosis. Occasionally, sclerosing or condensing osteitis (Fig. 10), which is a sequela of an inflammatory condition, may be confused with ossifying fibroma.

Periapical Cemental Dysplasia

Periapical cemental dysplasia (formerly known as cementoma, fibrocementoma, sclerosing cementoma, and periapical fibro-osteoma) is believed to be a reactive process. The origin of the lesion is unknown. The lesion is asymptomatic and usually is discovered during radiographic examination. Women, especially black women, are affected more often than men. The lesions typically appear in middle-aged individuals (mean age, 39 years). The mandible, anterior segment in particular, is far more involved than the maxilla. All the teeth in the affected area are vital.

Histologically, the lesion consists of a mixture of benign fibroconnective tissue, cementum, and bone. The ratio of these structures depends on the biologic age of the lesion. Newer lesions are naturally more radiolucent than older ones. The calcified material is arranged in trabeculae, spicules, and irregular masses. Osteoblastic and/or cementoblastic activity and rimming along the calcified islands are present. Mononuclear inflammatory cells may also be seen.

Radiographic Features. The radiographic appearance of periapical cemental dysplasia varies depending on the stage of the lesion. In the early stage, the lesion appears as single or multiple ill-defined radiolucencies at the apex or apices of the involved teeth. This stage of the disease simulates a periapical inflammatory process. In the second stage of development, the lesion will appear as a mixed radiodensity on radiographs. In the final stage, the lesion appears sclerotic, often completely calcified, and surrounded by a radiolucent rimming (Figs. 11 and 12). Periapical cemental dysplasia may appear as multiple separated lesions or they may coalesce and form a larger lesion (Fig. 12). See also the article by Weber elsewhere in this issue.

Florid Osseous Dysplasia

Florid osseous dysplasia is a rare condition that is believed to be an exuberant form of periapical cemental dysplasia. The two conditions share similar features, including patient age, sex, race, and histologic features. Florid osseous dysplasia is typically bilateral and may affect all four quadrants (Fig. 13). Occasionally, a simple bone cyst has been found in the affected tissue.[43]

Both of these conditions are innocuous, self-limiting, and need no treatment.

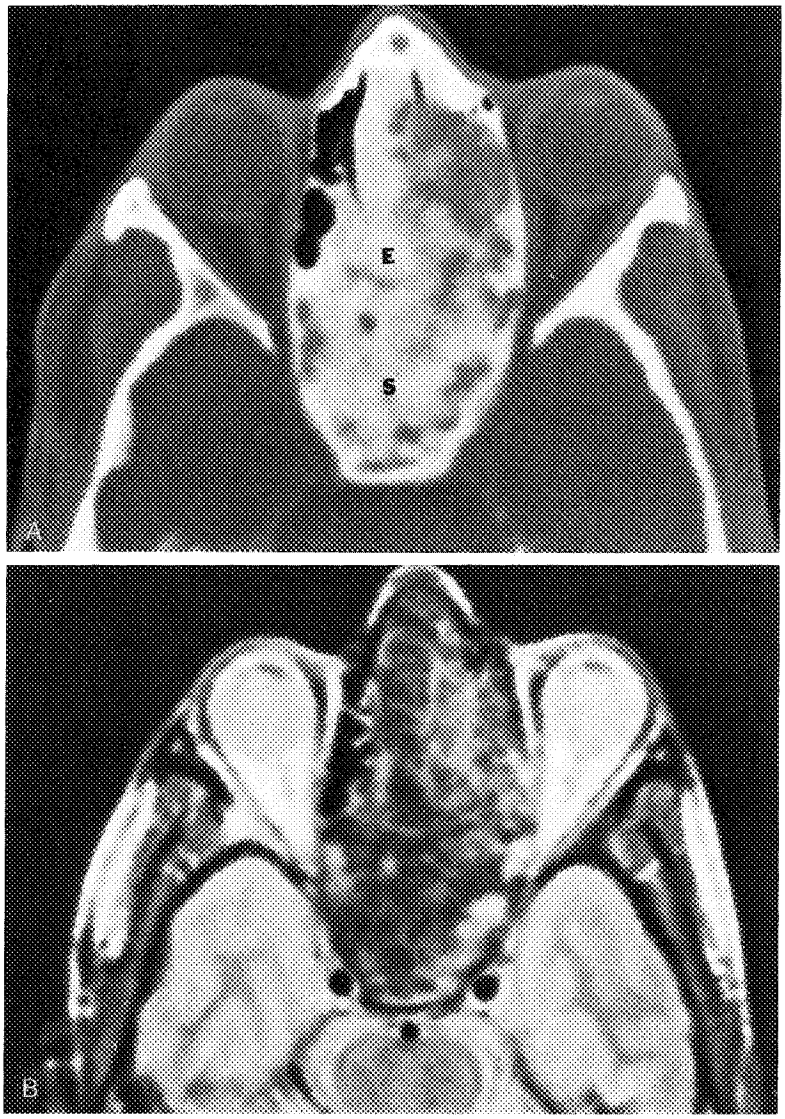

Figure 7. Presumed fibrous dysplasia. *A*, Axial CT scan shows involvement of ethmoid *(E)* and sphenoid *(S)* bones. *B*, Proton-weighted (proton density) axial MR scan shows heterogeneous signals characteristically seen in fibro-osseous lesions.

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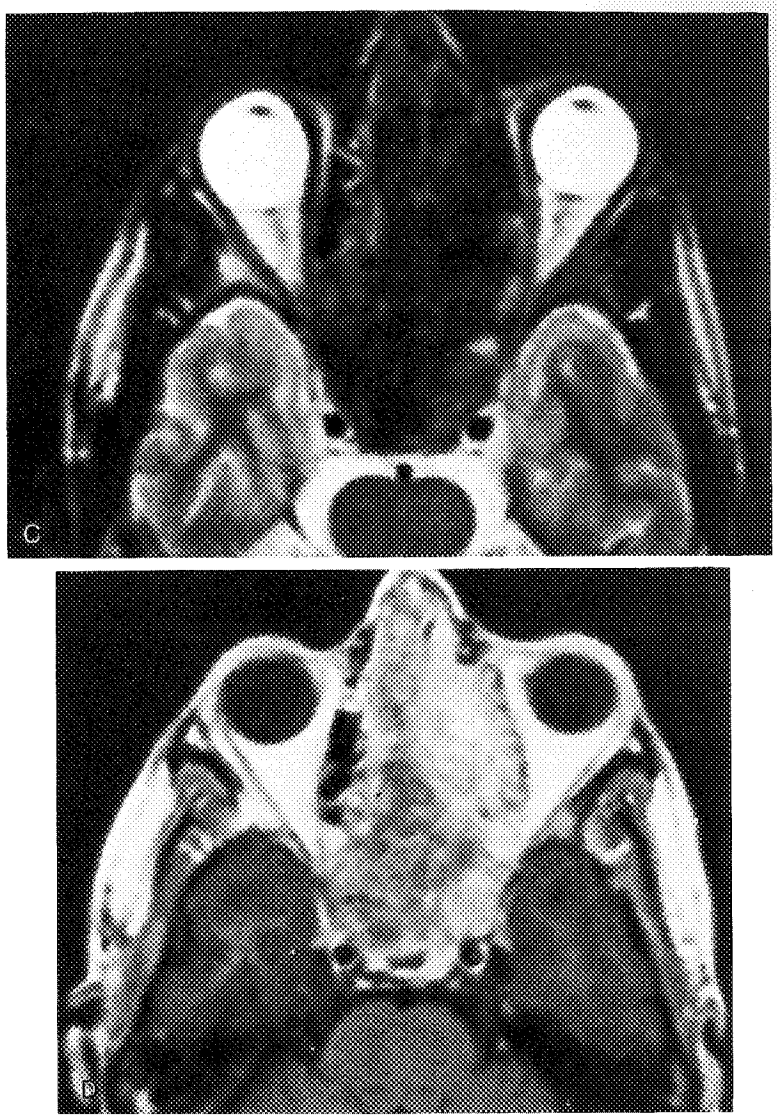

Figure 7 *(Continued). C,* T2-weighted axial MR scan shows marked hypointensity of ethmoid and sphenoid lesions. *D,* Post-gadolinium-enhanced T1-weighted MR scan shows marked enhancement within the lesions. (Courtesy of M. Mafee, MD.)

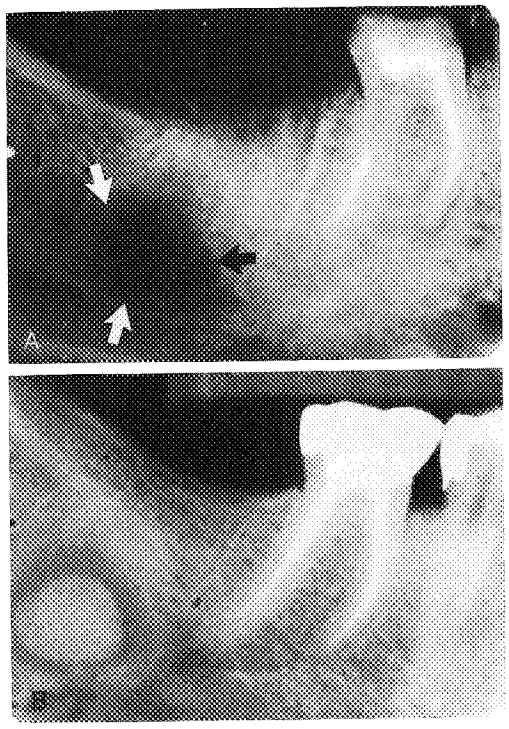

Figure 8. Periapical views of an ossifying fibroma. *A,* Radiolucent stage of the lesion *(arrows). B,* Late stage of the same lesion (4 years later). Notice marked sclerosis of the lesion with a peripheral rim of less radiodensity.

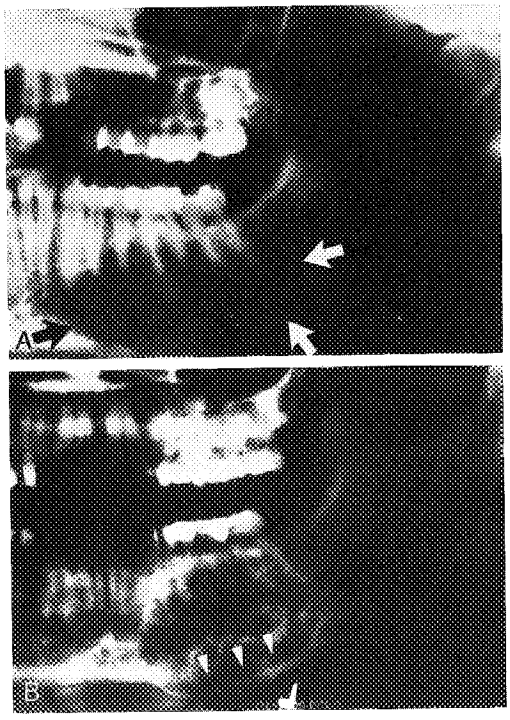

Figure 9. Early *(A)* and late stage *(B)* of a mandibular ossifying fibroma. *A,* Note radiolucent expansile lesion *(arrows). B,* Note some degree of irregular bone formation within the lesions and with inferior displacement of the inferior alveolar canal *(arrowheads).*

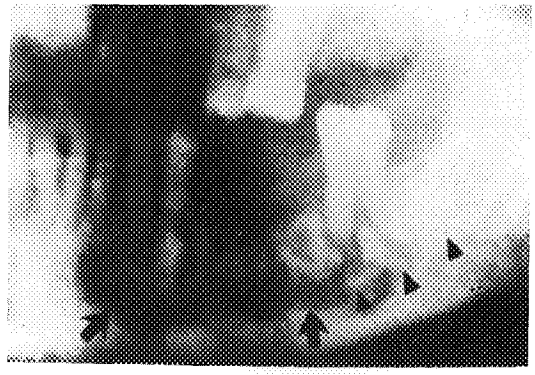

Figure 10. Panoramic view of the mandible in condensing osteitis due to advanced periodontal disease in a 60-year-old woman. Note ill-defined areas of reactive bone sclerosis *(arrowheads)* as well as areas of loss of bone density *(arrows)*. Differential diagnosis for this lesion should include florid osseous dysplasia and chronic osteomyelitis.

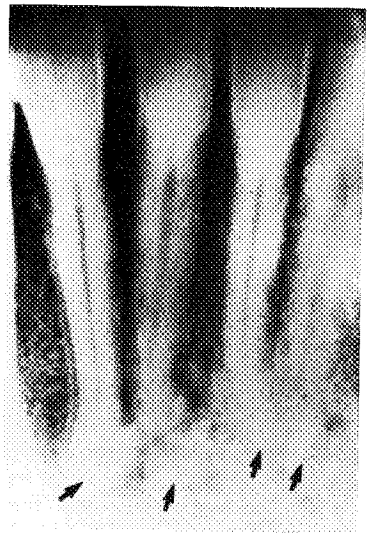

Figure 11. Late stage of a periapical cemental dysplasia. Note periapical radiodensities *(arrows)* with radiolucent rim. All the involvied teeth were vital.

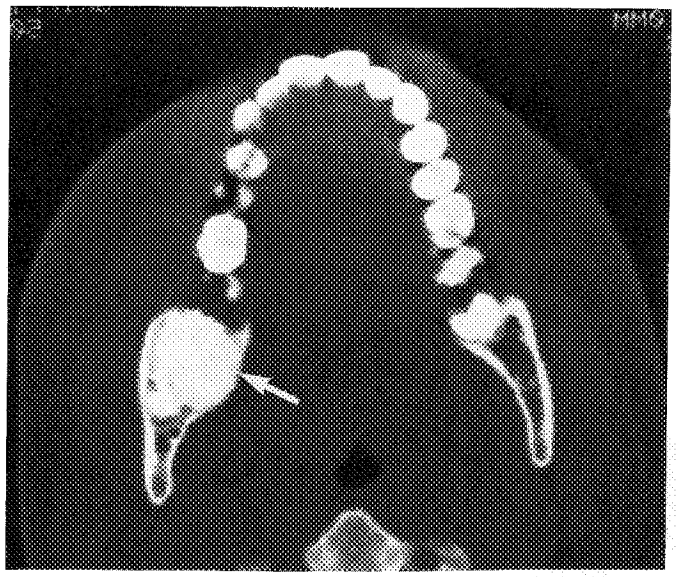

Figure 12. Cementoma. Axial CT scan shows a heavily calcified periapical mass *(arrow)*. (Courtesy of M. Mafee, MD.)

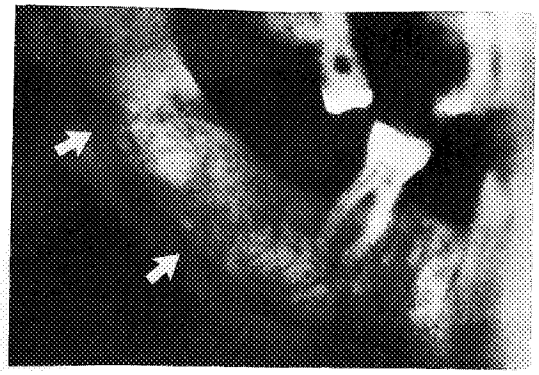

Figure 13. Florid osseous dysplasia in a 55-year-old woman. Panoramic view of mandible shows diffuse and ill-defined globular radiodensity. Note mandibular canal *(arrows)*. Differential diagnosis includes diffuse sclerosing osteitis.

Cementifying Fibroma (Cemento-Ossifying Fibroma)

Some oral pathologists include these cementifying fibromas in the spectrum of fibro-osseous lesions arising from the periodontal ligament.[24, 43] Clinically and radiographically, these lesions may be similar to ossifying fibroma (Fig. 14). The difference lies in the histologic features of these lesions: cementifying fibroma consists of cellular fibrous tissue with rare mitotic activity. Calcified elements in the lesion appear to be globular cementum (or an island of calcified materials surrounded by cementoid and cementoblasts). The difficulties in separating ossifying fibroma from cementifying fibroma has resulted in the term *cemento-ossifying fibroma* to encompass these identical tumors.

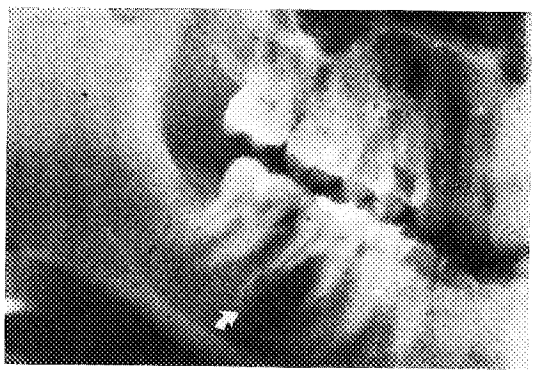

Figure 14. Cemento-ossifying fibroma. This asymptomatic lesion at the apex of the right mandibular first molar *(arrow)* was discovered during dental examination. Histologically, this lesion proved to be a cemento-ossifying fibroma. The involved tooth was vital. Note the well-defined radiolucency with mild distal root resorption.

DISCUSSION

Familiarity with various fibro-osseous lesions of the craniofacial structures is critical for proper imaging interpretation of these lesions. On differing diagnostic imaging modalities, these lesions may share similar features and characteristics, making it difficult for a definitive diagnosis.

Adequate clinical and paraclinical information, such as the patient's age, sex, location of the lesion, duration of symptoms, imaging characteristics, and histopathologic findings are necessary to reach an accurate diagnosis (Table 1).

Fibrous dysplasia is a developmental anomaly and affects children and young adults. Radiographically, these lesions often appear diffuse regardless of their morphologic stage. The borders are not readily distinguishable from the uninvolved surrounding bone, especially on conventional imaging. Histologically, in their classical form, bone formation is de novo, that is, they develop directly from fibrous tissue with less osteoblastic activity, and their maturation is arrested at the woven stage. Treatment must take a cosmetic approach, usually offered after puberty when the growth process is completed.

Ossifying fibroma, on the other hand, appears to be a fairly encapsulated benign neoplasm. With the exception of the juvenile variety, the patients are usually in the third or fourth decade of life. Radiographically, the tumor often appears well circumscribed and round. Histologically, it is more vascular, and bone formation is more lamellar. There is noticeable osteoblast proliferation, especially around the trabeculae. Surgical enucleation is an accepted treatment, although because of its aggressive nature and recurrence tendency, the juvenile form of this tumor requires total excision.

Cementifying fibroma has clinical and radiographic characteristics essentially similar to ossifying fibroma. The histologic findings of globular, small-sized acellular cementicle and/or cementoid structures are the differentiating elements.

Periapical cemental dysplasia and its exuberant counterpart, florid osseous dysplasia, are most likely reactive processes. The location of the lesion, the periapical region of the anterior mandibular teeth for the former conditions and the bilateral body of the mandible or maxilla for the latter, are quite characteristic of these lesions. Teeth in the affected areas are usually vital and nondisplaced. The periodontal spaces of these teeth are spared.

The differential diagnosis provided by the radiologists is invaluable for clinicians in preventing unnecessary procedures. Despite the clinical, radiographic, and to some extent the histologic similarities, there are diverse biologic behaviors ranging from simple reactive processes such as cemental dysplasia to aggressive and destructive lesions such as juvenile ossifying fibroma. Each lesion may need a different treatment approach.

Table 1. DIFFERENTIAL FEATURES AMONG BENIGN FIBRO-OSSEOUS LESIONS OF JAW BONES

Pathology	Age (decade)	Sex	Preferred Site	Radiographic Findings	Bone Expansion
FD	1st & 2nd	M,F	Maxilla	Ill-defined margin	+
OF	3rd & 4th	F	Mandible	Well-defined RL, RO	+
PCD	3rd & 4th	F	Mandible	Well-defined, multiple	–
FO	3rd & 4th	F	Mandible	Well-defined bilateral	–
COF	3rd & 4th	F	Mandible	Well-defined	+

COF = Cemento-ossifying fibroma, FD = fibrous dysplasia, FO = florid osseous dysplasia, OF = ossifying fibroma, PCD = periapical cemental dysplasia, RL = radiolucency, RO = radiopacity.

ACKNOWLEDGMENT

The authors thank Mahmood F. Mafee for his expert review of the manuscript.

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